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PHYTOPATHOLOGY

VOLUME XI NUMBER 2

FEBRUARY, 1921

THE BACTERIAL BLIGHT OF THE BEAN: A SYSTEMIC DISEASE

WALTER H. BURKHOLDER

The bacterial blight or bacteriosis is a common and widely distributed disease of the bean. Although at one time it was thought to cause but little damage, it has increased in importance in the last few years until now it is one of the serious diseases of the crop. The U. S. Plant Disease Bulletin for 1919 shows that during that year the bacterial blight was present in all the important bean-producing districts in the United States, causing an estimated loss of from 3 to 8 per cent of the crop in the states listed. In New York State, during the last three years, the disease has been found by field surveys to be of greater importance than the anthracnose.

The cause of the bacterial blight is well known from the classical work of Erwin F. Smith (8, 9), who studied and named the organism *Bacterium phaseoli*. Notwithstanding this work of Smith and the work of of Edgerton and Moreland (2), Muncie (5, 6), and others, there are many phases of the disease that are not well understood. The writer has been making a study of the disease as it occurs in New York State, and while the work is by no means completed, certain facts of such outstanding importance have been determined that it seems worth while to present them at this time. It is believed that the systemic nature of the bacterial blight, evidence of which will be presented, accounts in a large measure for the various anomalies met with in this disease. Unlike many of the parasitic plant diseases there are different symptoms depending upon a variety of conditions not clearly understood.

USUAL SYMPTOMS

In general the bacterial blight is thought to be a leaf and pod disease. It is on these parts of the bean plant that the most striking evidence

of the disease occurs. On the leaves the lesions first appear as small, water-soaked or light green, wilted areas. As the spots develop the tissue becomes dry and brittle with a yellow border, surrounding which there is frequently a paler green zone. One or more of these lesions may increase in size and involve a large part of the leaflet. No other disease of the bean produces on the leaf as large a necrotic lesion as does the blight; consequently this symptom is seldom confused with that of any other disease. In epiphytotics of the bacterial blight the foliage of most of the plants in a field may be affected, and from this, together with the whipping of the wind and rain, a very ragged appearance results.

The bacterial blight causes small, water-soaked spots on the pods, which gradually enlarge to irregular blotches. A yellowish-white incrustation, due to the bacteria oozing out in large masses and drying, frequently covers these spots. As the lesions become older, the tissue involved loses its water-soaked appearance and becomes dry and sunken. The color of the spots is often a brick red and later assumes a brownish tinge.

The bacteria on the pods readily work their way through the tissue and infect the bean seed. If infection takes place when the pods are young and tender, the seeds may rot entirely, or become so severely injured that there appears to be nothing left but the shriveled seed-coat. On the other hand, the bacteria may cause on the bean seeds only slight spots, in many cases similar to those produced by the anthracnose fungus. On white seeds the blight lesions are yellow and, when covering a considerable area, frequently varnish-like. When *Bacterium phaseoli* once gains entrance into the seed it persists over winter, and various attempts to kill it by seed treatment have been unsuccessful. In New York State, Red Kidney seeds two years old have been found to harbor viable bacteria, and in several cases seeds three years old when planted have produced blighted plants. This is contrary to the report of Rapp (7) from Oklahoma, who states that the blight organism is found to be dead in bean seeds two and three years old.

On the stem of affected bean plants various types of lesions have been observed to occur. In the seedling stage these lesions appear as small, water-soaked areas similar in appearance to those found on the pods. They may be distinguished from the anthracnose spots, as they are never so deep in the tissue, nor so dark in color. On the older plants the lesions appear as reddish dashes extending longitudinally with the stem. In these spots the tissue frequently is somewhat raised on the surface and split. The lesions on the stalks causing

the most damage to the plant have been referred to by the writer (1) and Muncie (5) as stem girdle. In this case the bacteria attack the stalk at the cotyledon node, or less frequently at the various other nodes higher up the plant. A red necrotic lesion is formed, which greatly weakens the stem and causes it to be toppled over very easily during a brisk wind. Fields have been observed by the writer where during a storm nearly 5 per cent of the plants were blown over in this manner. The varieties Red Kidney and Refugee appear to be very susceptible to this form of the disease.

UNUSUAL SYMPTOMS

Besides these more or less common symptoms occurring on the bean plant, a number of other manifestations of the bacterial blight may be found. Certain of these are fairly common in the bean fields of New York State, but since in some cases they occur on seedlings, they have been overlooked. The blight is never severe epiphytotically until near the maturity of the plants. In the early stages of growth of the host, however, certain lesions are present, the bacteria causing which act as sources of infection for the later spread of the pathogene. From all observations, these lesions arise from infection of the cotyledons due to the planting of blight-infected seeds. Possibly one of the earliest symptoms to be noticed is the appearance of the so-called "snake-heads," a name applied to the young seedlings when the growing tip of the plant has been destroyed and only the cotyledons remain. At times the snake-heads are able to send forth new shoots, but they seldom produce a large number of pods. Such injury has been attributed by Hawley (3) to the seed-corn maggot (*Phorbia fusiceps* Zet.) which is undoubtedly the cause in the majority of cases. On the other hand, in the spring of 1919 a great many injured seedlings were found on which no attacks of the maggots were in evidence. In many such instances bacteria were associated with the injury. The bacteria when isolated and used for inoculations produced a blighted condition in the bean plant, and were to all appearances *Bacterium phaseoli*. Moreover, on January 21, 1919, fifty White Marrow bean seeds showing blight lesions near the plumule end were planted in flats in the greenhouse. Ten of these produced typical snakeheads. Other experiments in which blight-infected seeds have been used have given varying percentages of seedlings with similar injury. It should be stated here that the writer also has seen *Thielavia basicola* Zopf and Rhizoctonia cause this injury to the young seedlings. In these cases the fungus was always in evidence.

Macroscopically it is impossible to determine with certainty the lesions on the cotyledons due to the blight bacteria. Many other organisms, both pathogenes and saprophytes, may cause like spots, in most cases all being a brownish color. Peculiar lesions, however, are produced on the primary leaves by *Bacterium phaseoli*. Here the leaf is marked as a mosaic, with small, angular, water-soaked areas over the surface. This is due to the infection of the islet tissue between the veins. Such lesions frequently do not develop, and the plant appears to throw off the disease. At other times the disease may appear on the young leaves as spots similar to those on the older leaves. After inoculating six small seedlings, February 27, 1920, in the greenhouse, by spraying the leaves with a water suspension of *Bacterium phaseoli*, the mosaic-like lesions were produced in all cases. The incubation period was five days. Later the spots on several of the leaves enlarged to the other type of lesion.

Another sympton of the bacterial blight that occurs in the seedling stage is the wilt. Occasionally this symptom may be observed in the field, and more frequently in the greenhouse where blight-infected seeds have been planted. A slight flagging of the leaves usually takes place, followed shortly by a drooping and general wilting. The stalk in most cases remains upright. On some seedlings one leaf will wilt before the other. In conducting inoculation experiments to determine the ability of *Bacterium phaseoli* to cause a seedling wilt, this one-sided wilting of the plant occurred in the majority of the cases. Such an uneven progress of the disease is due, no doubt, to the bacteria becoming established on one side of the plant only. It should be stated also that only a small percentage of the inoculations produced seedling wilt. Infections, however, frequently appear in the later development of the host plant, showing that external conditions governing the seedling wilt are not understood.

From the preceding paragraph it might be concluded that wilt is to be found only in the seedling stage. Such an assumption, however, is not true. In New York State during the last few years the wilting symptom of the blight occurred very generally in the White Marrow bean fields, especially after the pods had set. In some instances the entire plant died, while in others only a portion of the plant showed the disease. In the latter case, however, the wilting frequently spread until it involved the whole bean plant. The wilting symptom of the bacterial blight is one of the evidences of the systemic nature of the disease, and is treated below in more detail.

SYSTEMIC INVASION

During August of 1918, while examining several fields of White Marrow and Red Marrow beans in western New York, the writer found a number of apparently healthy plants producing blighted and water-soaked seeds. Further inspection of these bean plants showed that they were somewhat dwarfed, and a microscopic study revealed the fact that the vascular systems of the stalks were invaded by bacteria. No external lesions occurred on the leaves and pods. Since the bacterial blight caused by *Bacterium phaseoli* was found present in the field, it was considered probable that the blighted seeds were caused by this disease. Furthermore, about harvest time, clean appearing pods were found containing seeds with typical bacterial blight spots on them. Other parts of the plants also showed traces of the disease. Careful examination of these seeds revealed yellowish discolorations on the hilum, which indicated that the pathogene had entered the pod through the vascular system. Later, inoculation experiments conducted on bean plants with the bacteria isolated from individuals with infected vascular systems gave spots on the leaves and pods typical of the bacterial blight.

During the summer of 1919, careful observations were made of the development of the blight in the bean fields, and by these, together with inoculation experiments, the following points in the life-history of the pathogene were determined. From the primary infection of the cotyledons the bacteria readily gain entrance into the vascular system of the bean plant. The seedling wilt is a result of the behavior of the pathogene at this time. Due to some conditions not understood, bacteria rapidly increase, fill the xylem vessels, and a wilt soon follows. Upon microscopic examination the organisms will be found in great masses in the xylem vessels extending up the stem from the cotyledon node to the leaves. They also may be found extending down the stem about an inch below the cotyledons. In severe cases bacteria break through the walls of the vessels and spread into the nearby parenchymatous cells. That the pathogene causing seedling wilt may enter the stem from infected primary leaves as well as from the cotyledons seems probable, since in a number of cases the bacteria do not extend down the stem to the cotyledons. It should be stated here that the mosaic-like mottling of the primary leaves is due to the infection of the palisade cells and the mesophyl tissue, while the larger leaf spots, considered more typical of the blight, are caused primarily by the pathogene in the leaf veins. In the latter case the parenchymatous cells may be involved, but the rapid development of the lesion is due to the pathogene traveling in the xylem.

Due to some conditions not sufficiently understood, the bacteria at times enter the vascular system of the stem without causing a wilting of the young plant. Here the organism persists, causing a dwarfing of the plant, and on days of high evaporation producing an incipient wilting. Furthermore, on such plants a burning of the tips of the leaves has been observed to occur without the accompanying presence of the bacteria in the leaf. This last symptom undoubtedly is caused by the presence of the pathogene in great masses, plugging up the vessels and disturbing the transpiration of the plant. The White Marrow bean plant, which has an indeterminate habit of growth, fails to vine under such conditions. Few pods are formed, and the affected plants are dwarfed and mature earlier than the normal individuals. Such plants resemble somewhat those infected with the root-rot organism, *Fusarium martii phaseoli*. Consequently, without careful examination the two diseases may be confused.

Bacterium phaseoli infecting the vascular system of the host plant may behave differently under unlike conditions and in the different varieties of beans. In many instances, no external lesions or death of the plant parts occur until blossoming time, or even after the pods have set. The entire plant may wilt with the leaves and pods persisting on the plant, as frequently happens in the case of the White Marrow variety of beans. In the pea and kidney varieties, the leaves may fall and leave the clean, unspotted pods exposed.

Besides the wilting just described, other symptoms from the systemic infection occur on the plant. The bacteria may break through the xylem vessels and cause lesions to be produced on the leaves similar to those caused by local infection. First the tip or some other portion of the leaf wilts and later becomes dry and brown. These dead areas finally involve the entire leaf. On the stem the bacteria break through, producing reddish necrotic lesions. These appear as streaks that extend longitudinally with the branch or petiole on which they occur. At other times the lesions may not break through the surface, but remain as reddish-brown discolorations beneath the epidermis. From careful examination of the stem girdle, it appears that it also arises in some cases from the bacteria in the vascular system.

In the vascular infected plants the bacteria often extend not only throughout the parts above ground, but also into the root system. Here in the xylem vessels of the tap and lateral roots, great masses of the organism may be found as in the portion of the plant above ground. No external lesions, however, have been observed to be produced by the pathogene below ground.

One of the most important points in the behavior of the blight bacterium is its ability to enter the pods through the vascular system and infect the seeds without causing lesions on the surface of the pods. In entering the seeds through the vascular system, the pathogene frequently causes only a small, yellow discoloration at the hilum. This sign of the disease in colored seeds is not readily detected. In white seeds also, where normally there is a slight yellow marking about the hilum, the infection may be overlooked. Severe infection, however, produces on the seed-coat the yellow blotches so characteristic of the blight disease. This method of infecting the seeds without producing lesions on the pods is of considerable importance from the control standpoint. It immediately becomes evident that control cannot be accomplished through the selection of healthy pods. Such a plan has been recommended for the bacterial blight, since it proved successful in controlling the anthracnose disease. With the anthracnose, however, all infections are local. To be certain of securing bean seed free from the bacterial blight, healthy plants only should be selected.

It is very probable that in a large majority of cases the hilum-infected seeds carry the bacteria over winter. It is difficult for shriveled or undersized seeds or those with discolored spots on them to pass through a modern bean seed house, as such seeds are carefully gleaned by machinery or by the pickers. Many of the hilum-infected seeds appear normal in size, and only the slight yellow spot reveals the infection. Such seeds on planting may produce diseased plants.

This systemic infection of the bean plant by *Bacterium phaseoli* occurs, no doubt, wherever the disease is found. In New York State it has been observed frequently during the last two years. Jones (4) gives plates and descriptions which indicate that it is found in Ontario, Canada. Barss, in a conversation with the writer, stated that such plants occur in the bean sections of the Pacific Coast. This condition, however, may be overlooked readily, which doubtless accounts for its not having been emphasized before.

In the bean sections of New York State it is not this type of infection, however, that causes the greatest damage. Plants showing systemic infection occurring from the seed are, as a rule, in the minority. They do serve, nevertheless, as sources of inoculum for the epiphytotics of this disease that appear in August. In the fields, up to blossoming time, the bacterial blight is seldom an important factor, and is rarely noted. During the hot month of August, however, it frequently breaks forth. Possibly the temperature has some bearing on its severity at this time of the season. The mature leaves, too, readily become infected. The blight under these conditions spreads very rapidly over

a planting. Rain and insects appear to aid in the dissemination of the pathogene. Among the dry shell beans, the kidney varieties become more severely infected than either the pea or marrow varieties. A number of the garden varieties appear to be even more susceptible than the kidney.

EXPERIMENTAL WORK

A number of inoculation experiments have been conducted from time to time to determine the various points brought out in the preceding account. Some of the more typical ones are here recorded. On May 21, 1919, twenty-one White Marrow and Red Kidney plants about three weeks old were inoculated by injecting into the plants at the point of cotyledon attachment a water suspension of *Bacterium phaseoli*. In doing this a hypodermic needle was used and the stem pierced so that the vascular system was broken at one point. The inoculated plants were kept in a moist chamber for several days, after which they were placed in the greenhouse, where the temperature is usually high at that time of the year. The bacteria used in the experiment were isolated from a blighted White Marrow plant that had been grown in the greenhouse from an infected seed. After a week's time, symptoms of the disease began to appear, and within two weeks sixteen of the plants showed blight lesions, which in most cases were on the side of the plant where inoculations had been made. In all instances one or more of the leaves on a plant showed signs of spotting and wilting. The presence of bacteria was also discovered in the xylem and could be traced from lesion to inoculation court. This one of several similar experiments shows the ability of *Bacterium phaseoli* to travel through the vascular system from the cotyledon node to the leaves of a bean plant, and there produce lesions.

A further experiment to prove the systemic nature of the bacterial blight and also the ability of the hilum-infected seeds to produce blighted plants was conducted. On January 23, 1920, fifty Wells Red Kidney bean seeds that showed discolorations on the hilum but otherwise appeared normal were planted. The field from which the seeds had been secured had been severely infected with the blight, so that it was thought a large percentage of the discolored hila were due to *Bacterium phaseoli*. Only thirty-eight of the seeds germinated, but all of these grew past the seedling stage without any visible signs of the disease. By February 27, however, twelve of the plants showed symptoms of the bacterial blight. Four had wilted entirely. On the other eight the leaves flagged and died from time to time, and the bacteria in several cases broke out on the stem. On examining the infected plants the xylem

vessels were found to be filled with bacteria. The cells about the vessels had become infected also. On February 27, the stem of one of the plants was broken open and placed in a small amount of water, the bacteria being allowed to ooze out. This water was then sprayed over five Marrow bean plants, which were placed in a moist chamber for two days and then left in a greenhouse kept at a temperature of about 26° C. Five days later there appeared on the leaves of the inoculated plants small, water-soaked spots, which in about two weeks developed into the larger and more typical blight lesions.

When inoculation experiments were conducted with bacteria taken from pure cultures, negative results were very frequently obtained. It is thought that *Bacterium phaseoli* probably loses its virulence very rapidly in culture. In making inoculations with the organism from infected plants good infection resulted in the majority of cases.

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THE INCREASE IN RESISTANCE TO CITRUS CANKER WITH THE ADVANCE IN MATURITY OF CITRUS TREES

H. ATHERTON LEE[1]

The present paper advances the hypothesis that as citrus trees advance in maturity there is a gradual increase in their resistance to citrus canker. This hypothesis is based on field observations only, but it has been found to hold true in so many cases and under such varied conditions that it is advanced here in this form to aid in other problems of citrus canker investigations and to afford a better understanding of the disease to those who are not well acquainted with it in the field. The field observations which have given rise to this hypothesis are as follows:

At Singalong, Philippine Islands, nursery trees of calamondin (*Citrus mitis*) are grown; at the time of the observation these nursery trees were two years old and were very badly affected with canker, usually 75 or 80 percent of the leaves of each tree showing infections. Mature trees of the calamondin are grown nearby, subject to entirely the same conditions of sun, wind, rain and humidity and the same sources for infection; the writer does not recall and has no record of ever having seen a canker infection upon these mature trees or upon any such mature calamondin trees throughout the Island. The calamondin is generally accepted as entirely free from canker throughout the Philippines. At Lamao, Philippine Islands, nursery trees of the calamondin are grown and are found to be very abundantly cankered; at the time of the observation these trees were a little more than one year old. In the same planting with these nursery trees occur mature calamondin trees six to seven years old; such mature trees have never shown a canker infection although other orchard trees nearby of more susceptible varieties, as well as the nursery trees, present abundant sources for infection.

Two-year-old trees of the mandarin orange (*Citrus nobilis*) are grown in nursery rows at Singalong, P. I.; as found there they were at the time of the observation very susceptible to canker, showing a large percentage of leaves with one or more infections. Nearby mature trees of the same species showed no citrus canker although exposed to the same climatic conditions and same sources of infection. Trees of the mandarin orange less than two years old were observed at Luta, in the Philippines, to be commonly affected with citrus canker; mature trees of the mandarin orange but a few feet from these young trees showed no infections from canker although exposed to entirely similar conditions of air, wind, etc., and having the same constant sources of infection.

Year-old trees of the Chu Kaa (Vermilion orange *C. nobilis*) growing at Swatow, China, were observed to be very badly cankered. The orchard plantings of mature trees of the Chu Kaa are very extensive around Swatow; although there are abundant sweet orange trees which afford sources for infection, no citrus canker infection was

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observed upon a single mature tree of this variety. Many visits have been made to the Swatow orchards at different seasons to determine this point. Many of these orchards have the susceptible year-old nursery trees interplanted among the mature trees, providing abundant sources for infection; in such cases the sun, wind, rain and humidity conditions for the young trees and the mature trees are entirely identical.

The Pong Kaa, a mandarin orange variety (*C. nobilis*) also grown at Swatow, gave the same observations. Year-old trees of this variety in many instances showed cases of defoliation resulting from canker infections; the mature trees, four or more years of age, are grown with the young trees interplanted, and show no infections whatsoever. The Pong Kaa when mature may safely be called resistant and no difficulty results from citrus canker.

Young pummelo trees (*Citrus maxima*) less than two years old are grown at Lamao and are there found to be very susceptible. As many as 90 or 95 percent of the leaves will sometimes show infections. Mature pummelo trees are grown at Lamao and throughout the Philippines. Such trees, although for the most part slightly susceptible to canker, are never very seriously affected; canker upon them causes negligible injury. Cases have been observed where such mature trees were exposed to the same climatic conditions and sources for infection as the young severely affected nursery trees, although the mature trees would remain but slightly affected.

Young nursery trees of the "Hirado Buntan," a Japanese pummelo variety, were observed at Nagasaki, Japan, to have considerable canker; at the time of the observation these trees were one year old. Under the same conditions in the same place, mature trees of the "Hirado Buntan" remained entirely free from canker; the "Hirado Buntan" is regarded as a resistant variety in Japan which may be safely grown without loss from canker.

Trees of the lemon varieties (*Citrus limonia*) less than one year old were observed at Okitsu, Japan, to be very severely affected by canker. In some cases killing of twigs resulted from girdling by canker. Mature trees four to six years old of the same varieties were grown nearby and although exposed to the same climatic conditions and sources of infection showed little or no citrus canker.

Year-old trees of the "Owari Unshiu" (*C. nobilis* var. *unshiu*) were observed in nursery rows at Okitsu and a considerable amount of canker infection was to be seen upon them. Mature orchard trees of the same variety nearby, however, showed not a single infection. The same observation on "Unshius" has been made near Kobe. These observations hold good throughout Japan, that is, "Unshiu" trees in the nurseries may frequently show an abundance of infection whereas it is very difficult to find infections on mature "Unshiu" trees.

Young trees of the "Natsu-daidai" (*Citrus aurantium* subsp. *intermedia* var. *natsu-daidai* Tanaka) were observed in nursery rows at Okitsu; as grown there they showed a considerable amount of infection with citrus canker. Mature "Natsu-daidai" trees in an orchard nearby were almost entirely free from canker. The "Natsu-daidai" is grown very widely throughout Japan and very little foliage infection is to be found upon mature trees of this variety.

Seedling trees of *Poncirus* (*Citrus*) *trifoliata* were observed in nursery rows near Kobe, Japan, and were very well infected with citrus canker. Three large mature trees of the same species grew at one side of the nursery and not a single infection could be found upon them. The mature trees were exposed to the same conditions of light, wind, rain and humidity and the same sources of infection as were the nursery trees. *Poncirus trifoliata* is grown very largely for use in hedges in Japan and infection of such large mature hedges is not at all common.

Several other similar cases may be recalled but have not at the time been definitely recorded. The converse, that is mature trees susceptible while young trees under same conditions of exposure have remained free from canker, has never been observed by the writer.

Field observations such as these cannot be taken as yielding the definite conclusion that trees gain in resistance to citrus canker with maturity. The observations are, however, apparently sufficient to warrant being used to formulate a working hypothesis which may be of value in other problems of citrus canker investigations. To summarize this hypothesis and explain more clearly: Citrus trees of the more resistant species (*Citrus nobilis*, *Citrus mitis*, etc.) may often show great susceptibility to canker when young, while with increasing maturity they become but slightly susceptible or entirely free of citrus canker. Although this applies more noticeably to the resistant species it is apparently true also for the more susceptible species, such as the grapefruit (*Citrus maxima*), the lime (*Citrus aurantifolia*) and the sweet orange (*Citrus sinensis*). Such species although showing abundant infection even when mature, do not often show the twig killing and never the complete defoliation which very frequently occur on nursery trees. The wording as follows therefore, is thought to be entirely safeguarded while at the same time covering all cases; the hypothesis is advanced on the foregoing evidence that as citrus trees advance in maturity there is a gradual increase in their resistance to citrus canker.

There are of course many complex developments which might occur within the tissues to create a greater resistance to canker with maturity. There are other more simple factors which might also have the same tendency to increase resistance with the aging of the trees. The quicker response of young trees to growth stimuli may be taken as one such simple factor which contributes to some extent to the greater susceptibility of young trees. To enlarge upon this: In periods of warm, wet weather favorable for canker development, growth will also be stimulated; on young trees the effects of such stimulation is to put out new growth very quickly; on old trees the effects of the stimulus are shown more slowly and often new growth will not appear until the period of climatic conditions favorable to canker infection has passed. New growth of course in most varieties and species is essential for canker development. In other words, the climatic conditions which are a stimulus to growth of citrus trees are also in a great many cases favorable for the dissemination and development of citrus canker. The old trees exhibiting growth slowly following these stimuli, very frequently will delay the new growth enough to avoid the conditions favorable to canker development. The young trees responding immediately to

growth stimuli cause the new growth in most cases to be subjected to climatic conditions favorable to canker development. This is but one factor which may contribute to the greater resistance of older trees. Undoubtedly there may be several factors more or less complex, all contributing to cause mature trees to be less susceptible to canker than the younger trees.

The working hypothesis advanced above should be kept in mind in discussing the resistance and susceptibility of Citrus species and varieties to citrus canker. In formulating ideas as to the susceptibility of a variety, it would seem well to check all such observations upon young trees with observations upon mature trees.

Another conclusion seems noteworthy. The difficulty experienced with citrus canker upon "Unshiu" varieties in the gulf states has been rather considerable in past years. It would seem probable that with the increasing maturity of "Unshiu" orchards in those states, that greater security from citrus canker can be felt and that a condition similar to that in Japan may be arrived at; that is, that even should sources of infection arise the mature "Unshiu" orchards will have little difficulty in remaining free from citrus canker.

It would seem also from the knowledge of the differences in the behavior of young plants compared with old plants, that all statements of injury caused by citrus canker to be of the greatest value should be coupled with the information as to the age of the tree as well as with the observations on the growing conditions, weather conditions and such other data.

FURTHER OBSERVATIONS ON A BACTERIAL ROOT AND STALK ROT OF FIELD CORN

H. R. Rosen

With Four Figures in the Text

INTRODUCTION

In a previous publication (3) the writer called attention to a field corn disease which he attributed to bacteria. Since then additional field and laboratory studies have been made involving observations not previously reported. It seems desirable to present further studies at this time, since this disease is apt to be confused with others which show somewhat similar symptoms, and since the previous account of this disease was merely preliminary in nature. This paper will be confined to a discussion of disease symptoms, the extent of damage, varieties attacked and artificial infections.

SYMPTOMS OF THE DISEASE

In the former publication (l. c.) it was noted that the disease manifests itself as a rotting of the roots and of the portion of the stalk to which they are attached and that individual roots showed brownish, dead areas, especially on the parts immediately joining the stem. It was likewise noted that the interior tissues of the lower nodes showed brownish discoloration and that this tissue was either dead or in a process of disintegration. Dark-brown spotting of husks, leaf blades and leaf sheaths was also noted. The disease has now been noted in other localities in Arkansas and similar symptoms, as well as others not previously described, have been observed. Most of the symptoms resulting from natural infections have been checked up by artificial infections which will be described later. The worst cases of natural infection were noted at Burdette, Arkansas. In this locality extreme cases of infection were found at the lower nodes, especially that immediately above the soil line. See figure 1. The tissues attacked showed marked decay and disintegration, appearing as a soft, dark-brown to blackish, water-soaked mass with a very strong odor of decay and involving the whole thickness of the stalk which at this point is considerably shrunken and collapsed. In the shrunken areas bacterial ooze in the form of

grayish-white slime may be observed. In such cases the stalk bends over and finally comes to rest on the ground. See figure 2. At times the attacked plant sends out secondary stalks from the portion below the attacked parts and such growths under favorable conditions may produce good-sized stalks. Often, however, the disease has apparently entirely killed the plant so that hills are represented by barren stalks lying on the ground.

FIG. 1. EXTREME ROTTING OF THE LOWER NODES OF JOHNSON COUNTY WHITE CORN. (Reduced.)

FIG. 2. STALKS FALLEN OVER AS A RESULT OF ROT AT THE LOWER NODES
Photographed at Burdette, Arkansas

Under conditions of high humidity and high temperature the decay progresses rapidly through the entire thickness of the stem, the attacked tissues collapsing and finally drying into a mass of shredded, easily disjointed fibers, remnants of the fibro-vascular bundles. Often, however, only portions of a node are found attacked, the decay varying from a dark-brown, water-soaked rot to a light-brown, dry rot, depending upon atmospheric conditions and upon age of infection. Various sized spots, stripes and blotches are also produced on stems, leaves and husks. Leaf spots are very common in the state, but most of these are of the type recently described by Durrell (1). Where nodal infections are abundant the sheaths surrounding these nodes are likewise attacked and artificial infections have been produced on sheaths, as well as various other parts of the corn plant closely resembling the natural infections here described.

All the writer's observations of this disease in the field were made on corn that had grown to good size. Attacks on seedlings and on corn at various stages of development were observed in artificial infections on material growing in a greenhouse. The localized rotting of lower nodes described for full-grown corn was also obtained on young seedlings six inches high by means of inoculations. See figure 3.

Rots produced by various fungi including Fusarium spp., *Diplodia zeae*, Verticillium and others are apt to be confused with the bacterial disease.[1] Disintegration of tissues occurs with Fusarium rots but ordinarily the lower nodes showing internal browning do not collapse and the falling over of stalks is not generally due to any localized rotted area brought about by attacks of Fusarium. Pammel, King, and Seal (2) present excellent photographs of Fusarium disease of corn and these correspond to the type of Fusarium injury found in this state. The only other disease known to the writer which may possibly be confused with this bacterial disease is Stewart's disease of sweet corn, but this, like Fusarium, is systemic, producing no localized infections comparable to those here described and is easily recognized by the yellow bacterial ooze observed in the bundles when the stalk is cut open.

FIG. 3. Sweet cornseedling, variety "Nectar," showing lower nodal rot resulting from artificial infection.

VARIETIES ATTACKED AND AMOUNT OF DAMAGE

The disease has now been located in several counties of Arkansas. Most of the corn grown on the farms of the Three States Lumber Company, Burdette, Arkansas, which has about 500 acres in corn, is Johnson County (white). By counts of diseased stalks per hundred, counting several hundred stalks per field, ten to thirty per cent of this corn was diseased. Quite often the disease appeared in contiguous hills of a

[1] Valleau (4) apparently questions whether there is a bacterial rot of corn. He says: "Seeds from five ears of a yellow corn grown in Arkansas were found to be badly infected with *Fusarium moniliforme*." Then he presents the following footnote: "These seeds were obtained from the farm of E. B. Whitfield, of Bauxite, Arkansas, on whose farm Rosen made his observations in regard to a bacterial root rot of corn." No comment need be made beyond stating that the "Gamble" variety of corn which was the one upon which the first studies of the bacterial disease were made is a white corn. Ninety-five per cent of the corn grown in Arkansas is white corn. Durrell (1) likewise assumes that there is no true bacterial disease of field corn and that the writer was in error in assigning certain leaf spots to pathogenic bacteria.

row, although it was also found more or less scattered. Some of the land had grown corn the year before, while some had not grown corn previously. The practice here has been to allow the stalks to stand in the field over winter and to plow them under the next spring. Besides the Johnson County corn the following fifteen varieties were found attacked. Reid's Yellow Dent, St. Charles Yellow, Boone County White, Boone County Special, Webb Watson, U. S. Selection 77, St. Charles White, Neal Paymaster, U. S. Selection 201, Biggs, Mosby, Hastings, Surecropper, Mexican June, and Huffman. Concerning the disease on these varieties, Mr. H. A. York, a graduate in agriculture from the state university, writes as follows: "The disease is found on all of them and. . . . . is worse in the lowest grounds." A white corn, known by the local name of "Gamble," and a variety of sweet corn known as "Nectar," both used in artificial inoculation experiments, were found to be susceptible. See figure 4.

ISOLATION AND INFECTION EXPERIMENTS

Since the disease was first described by the writer the pathogen has been isolated fifteen times. These isolations were from various tissues including roots, lower nodes including both the wet rot and the dry rot, leaves and husks.

FIG. 4. ROT FROM INOCULATION AT LOWER NODE OF "GAMBLE," FIELD CORN.

The pathogen is rather easily isolated. It is a rapidly growing organism, well separated colonies appearing within twenty hours on a +1.0 peptone-beef-agar poured plate kept at 25° C. Within forty-eight hours all colonies will have appeared. In case of good-sized tissue which can easily be cut open, as at nodes, isolations are attempted by using a flamed, sterilized knife and removing bits of the internal tissue and in case of leaf tissue or of nodes in seedlings the material is first surface sterilized. Details of these isolations will appear elsewhere.

The writer in the previous publication (3) reported artificial infections on corn roots and on leaves of corn and of oats. Numerous other infections have been obtained since then, involving more than 100 growing plants. Most of these were conducted in a greenhouse, a few in the field and some in the laboratory. Two methods were used, one in which the organism was smeared on the tissues by means of a sterilized platinum loop, the tissue being previously moistened with a spray of sterilized

water, and the other in which a few cubic centimeters of sterilized water is poured into an agar slant containing a bacterial smear, the smear thoroughly distributed in the water by means of a sterilized rod and the watery suspension of bacteria sprayed on the plant by means of a sterilized atomizer. When the plants were small enough to go under a bell-jar, they were kept forty-eight hours under the jars and then taken out and put on benches in the greenhouse. All plants were grown in six- or eight-inch pots and all belljars were wrapped with paper to prevent admission of direct sunlight. When plants were too large to go under a bell-jar the moistened tissue was smeared with the bacteria and moistened cotton tied around the smear. This was the method also employed in the field with full-grown plants.

Infection can be obtained with or without wounding and apparently on any part of the growing corn. The period of incubation varies from two to four days, the average being about three days. As a rule leaf infection, through stomata or water-pores, appears within forty-eight hours after inoculation ,while nodal infections take about twenty-four hours longer to appear. It will be recalled that aerial roots as well as all lateral roots in corn are of endogenous origin, breaking through the overlying tissue before reaching the surface. This ruptured tissue invests the base of the roots in the form of collar and it appears that infection begins at these natural wounds. Artificial infections have been produced on seedlings at these collars. The old leaf sheaths which envelope the base of the stalk and which under normal conditions die and disintegrate may also serve as infection points. In the former publication (3) the writer has already reported infection by means of root hairs.

A report of the morphology of the pathogen, its cultural reactions, identity, etc., will be left for a future paper.

SUMMARY

The bacterial disease of field corn appears as a localized rotting of roots and lower nodes of the stalk, and as spots on sheaths, blades, and husks.

Seventeen varieties of field corn and one variety of sweet corn are known to be subject to the disease. In one locality the extent of damage in mature corn, resulting in the death of the stalks, varied from ten to thirty per cent.

Fifteen series of isolations revealed a white, rapidly growing bacterium.

Artificial infections on more than 100 plants, resulting in nodal rot, root rot and leaf spot, are reported.

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METHODS OF DIRECT INOCULATION WITH DAMPING-OFF FUNGI

ANNIE E. RATHBUN[1]

WITH THREE FIGURES IN THE TEXT

Until the spring of 1919 Hartley (1) and his assistants had inoculated coniferous seedlings by adding the inoculum to the soil instead of by applying it directly to the seedlings. While this method of inoculation indicated great variability in the relative virulence of the different strains of fungi, there was a possibility that part of this relative virulence was due to the inability of some of the strains to maintain themselves saprophytically in the soil until the seeds had begun to germinate. Therefore, in order to eliminate this possibility, direct inoculations of both stems and roots were undertaken. The addition of the inoculum to the soil had also caused considerable germination loss but had not demonstrated whether the fungi could attack dormant seeds as well as the radicles immediately after their emergence from the seed-coats. For the purpose of ascertaining at what stage of development the fungi cause germination loss dormant seeds and radicles that had just emerged from the seed-coats were directly inoculated. During the progress of the above-mentioned experiments several new or modified methods were developed.

DIRECT INOCULATION OF STEMS

Inoculation at soil surface. The seedlings for direct inoculation experiments were grown in sand in small pots in the greenhouse and were inoculated at an age when the seed-coats were still clinging to the cotyledons. In the earlier experiments the inoculum was placed on the soil surface in contact with each seedling, which was marked by a tooth-pick at the time of inoculation. A tooth-pick was also used to divide the pot into halves in case it was desirable to use two kinds of inoculum—for instance, rice and agar (figure 1). The agar inoculum consisted of approximately 4 mm. squares. These were obtained by placing a 6–8 cm. square of 4 mm. coördinate paper, the lines of which had previously been accentuated, beneath a Petri dish culture and cutting along these lines.

[1] The writer wishes to acknowledge the great assistance of Dr. Carl Hartley in the development of these methods.

Inoculation platforms. The practise of placing the inoculum at the soil surface was soon discontinued in favor of the inoculation platforms (figure 1). The manufacture of these was very simple. Equilateral triangles with sides 3/8 of an inch long were cut from library cards. Tooth-picks were inserted through holes made in the center of these. An ink or blue pencil mark about 3/8 of an inch below the platform indicated how far the tooth-pick was to be pushed into the soil.

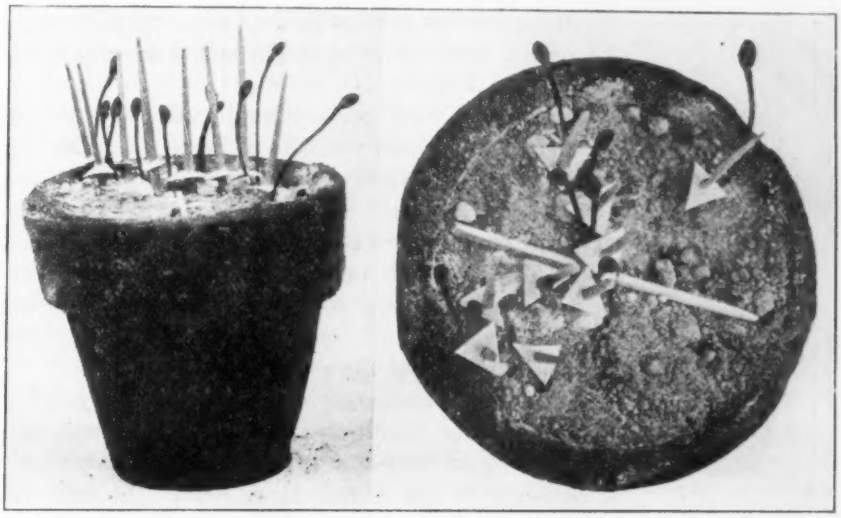

FIG. 1. TOP AND LATERAL VIEW OF POT SHOWING INOCULATION PLATFORMS

An inoculation platform was placed beside each seedling and upon it in contact with the stem of the seedling was placed the inoculum. Very often one platform held the inoculum for two or three seedlings.

This new method of direct inoculation was better than the first because it prevented the fungus from growing from one seedling to the other through the soil, and if the seedlings were examined daily, the exact point where damping-off began could be readily ascertained. This method has been used with apparent success in a rather large series of inoculation experiments.

CELLULOID CONES AS DAMP CHAMBERS

Modifications of Hubert's (2) celluloid cylinders were employed to provide damp chambers for these experiments. They consisted of truncated cones 5 inches high and 2½ inches in diameter at the base.

They were manufactured by the same method and from the same grade of celluloid—the thinner of the two grades of transparent celluloid such as is used for windows in automobile tops—that Hubert employed. "The edges" were "fastened together by means of acetic ether (ethyl acetate) applied by dipping a camel's hair brush in the liquid and running it quickly along the edges to be cemented." The two edges were kept firmly pressed together until dry (figure 2).

For use with pots, cones were much better than cylinders, because it was comparatively easy to get a tightly fitting cone without manufacturing one for each individual pot, and they could be nested and stored or transported in much less space.

FIG. 2. CONE DAMP-CHAMBER

The top of each cone was plugged with cotton wet in boiled water—moisture being furnished in this manner instead of by placing a piece of cotton inside as Hubert did. In case the sand itself became dry the pots were set in boiled water in sterilized pans. This indirect method of watering was necessary because overhead watering would have necessitated the removal of the cones, washed away the inocula, and moved the platforms.

As soon as the experiment no longer required damp chamber conditions the cotton was removed from the top of the cones. The cones still minimized, although to a less extent than before the removal of the cotton, the danger of contamination from such fungi as Fusarium and Botrytis which spread through the air. The use of boiled water prevented infection from water-borne fungi. As an additional precaution against accidental contamination each damp chamber was sterilized at the beginning of each experiment with a strong solution of formalin or mercuric chloride.

DIRECT INOCULATION OF TAP ROOTS

At the same time that some of the direct stem inoculations were being carried on, the tap roots of young seedlings were also directly inoculated. Three methods of inoculation have been tried with apparent success in a rather limited number of experiments.

The first and best method was to cut away one-half to two-thirds of a Petri dish culture; then to place the seedlings in the Petri dish in such a way that the roots were in contact with the inoculum while the stems merely touched the glass (figure 3).

A second method was to grow the fungi on rice mush in Erlenmeyer flasks. Several grains of the rice and the fungus growing upon them were transferred to the bottom of a sterile Petri dish. Then the seedlings to be inoculated were laid in the Petri dish with the root of each seedling in contact with the inoculum at one point while the remainder of the root and the stem rested against the sterile glass. With this latter method the seedlings showed a slight tendency to wilt.

In order to overcome this tendency the bottom of each Petri dish was lined with moistened filter paper before the inoculum was transferred to it. Otherwise this method did not differ from the second.

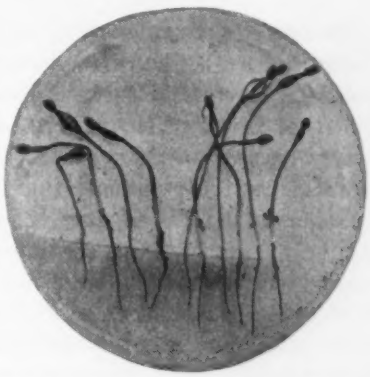

FIG. 3. INOCULATION OF TAP-ROOTS

Checks for each of these methods consisted of seedlings inoculated with sterile rice or agar. The fact that the check seedlings usually remained healthy seemed to prove that the root-rots were really caused by the introduced fungi and not by physiological changes brought about by the removal of the seedlings from the sand in which they had been grown.

DIRECT INOCULATION OF DORMANT SEEDS AND RADICLES JUST EMERGED FROM THE SEED-COATS

In order to ascertain whether or not damping-off fungi attack dormant seeds, it was necessary to germinate the seeds under conditions that made it possible for the observer to distinguish the ungerminated seeds from seedlings which were killed immediately after the emergence of the radicles from the seed-coats. Inoculation of the soil proved entirely useless for this purpose and a few small preliminary trials demonstrated that sowing seeds broadcast over a poured plate was not a good method because germination was poor even in the controls. Therefore, two hollow rectangles about $\frac{5}{8}$ of an inch wide and 2 inches long were cut in the interior of each Petri dish culture. Coördinate paper in which rectangles of the desired size had been drawn served as the pattern. The seeds were arranged along the perimeters of these rectangles with one side of each seed in contact with the inoculum. This method was better than sowing the seeds broadcast because (1) it did not smother the seeds, (2) it inoculated the seeds less heavily; (3) it made it easier to count the number of germinating seeds.

By this method radicles which had just emerged from the seed-coats were inoculated. If the seedlings were not attacked at this stage they generally continued to grow in the Petri dish, developing stems and cotyledons. The radicles which had just emerged from seeds previously germinated on filter paper were also inoculated by placing them in contact with rectangles of agar inoculum in sterile Petri dishes.

SUMMARY

The results of the methods described above will be published in detail elsewhere. (1) The direct inoculation of stems indicates that there is considerable variability in the relative virulence of the different strains and that a few of them were formerly considered non-parasitic apparently because of their inability to live in the soil. (2) The inoculations of tap-roots indicate that, although the methods should be tried out more extensively before definite conclusions are drawn, good results can be secured by their continued use. (3) The experiments with dormant seeds apparently confirm the assumption that by far the greater part of the germination loss is caused by the attack of radicles which have just emerged from the seed-coats rather than by attack of dormant seeds.

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BRIEFER ARTICLES

SOFT ROT OF PEPPER FRUITS

S. G. Lehman

During the rainy weather of July of the present year, a new disease of cultivated sweet peppers (*Capsicum annuum* var. *grossum*) appeared in the writer's garden at West Raleigh. As examination of available references indicates that this disease has not been described in pathological literature, it seems desirable to make a record of the trouble at this time.

The variety of pepper on which the disease appeared is a hybrid between the commonly cultivated forms of *C. annuum grossum* known as the Chinese Giant, and the pimento, the plants having been produced by Mr. L. R. Detjen, now associate horticulturist of the Delaware Agricultural Experiment Station. The disease has been found only on fruits, and natural infection has always occurred at the blossom end of peppers not more than six or eight inches above the ground. The trouble does not promise to become of much consequence economically as comparatively few fruits have been attacked.

The disease appears first as a small water-soaked spot at the point of inoculation. The infected area enlarges rapidly, becoming soft, watery, slightly sunken and somewhat lighter in color in contrast with the surrounding deep green uninvaded tissue. The parasite soon pervades the particular locule in which infection has occurred, and, within a period of four or five days, permeates the entire fruit, causing a soft, wet rot, with a brown surface discoloration. The epidermis remains unbroken and the discolored and misshapen fruit clings to the plant for several days. When infected fruits are kept in moist chambers, the fungus comes to the surface only where the epidermis has been broken, and at these places an abundant growth of white cottony mycelium is produced quite free from fruiting structures of any kind.

If a small portion of the infected area of the pericarp is crushed under a cover glass, the cells easily separate, and it can be seen that the tissue is permeated by an abundant mycelium of a phycomycetous nature. Pure cultures of the invading organism are easily obtained by sterilizing the unbroken surface of an infected fruit with mercuric chlorid solution and planting small pieces of invaded tissue in Petri dishes containing Czapek's nutrient solution solidified with agar-agar. An abun-

dant growth of sterile, non-septate mycelium is produced within a few days. When bits of infected tissue are put into water, in a watch glass kept at room temperature, branches of the mycelium grow into the water for a distance of a few millimeters and begin the formation of oogonia and antheridia within six or eight hours. This fruiting period continues for four or five days. At the end of this time, great numbers of oospores are present in and around the host tissue.

The mycelium branches irregularly, is non-septate, and finely granular when young, the hyphae having a diameter of 4–4.5 microns when two or three hours old. Old hyphae are coarsely granular, irregularly septate, and from 4–8 microns in diameter. Oogonia are globular, thin-walled, hyaline, and have a diameter of 16.5–23.7 microns (commonly 20.5–22.5 microns). Oospores are spherical, and they have a smooth light-brown wall which is often 1.5 microns thick. Their diameter is 12.4 to 18.4 microns. A rest period of considerable length is required before germination.

Antheridia are of two kinds, namely: stalk antheridia and branch antheridia. The stalk antheridium is a single cell which adjoins the oogonium and is formed by the production of a septum near the distal end of the oogoniophore. The branch antheridium consists of an enlarged cell at the end of a hypha which commonly, but not invariably, arises as a branch of the oogoniophore. Branch antheridia are club-shaped, possess a diameter of 6–6.5 microns and adhere closely to the oogonial wall. Fertilization tubes were readily seen in connection with both types of antheridia. Notwithstanding attempts to produce them by growing the fungus on animal tissue as suggested by Butler (2), no zoosporangia have been observed.

On animal tissue such as ants and flies, a profuse vegetative growth is produced, but no fruiting occurs except when the host tissue is immersed in water. Very few septa are to be found apart from oogonia and antheridia. A single hypha may possess diameters varying from 4–8 microns, measurements of adjacent portions frequently differing by 1–1½ microns. Oogonia and oospores are somewhat larger than corresponding structures produced on vegetable host tissue.

Attempts to accomplish artificial inoculation of healthy fruits were made in a number of ways. Pieces of pepper tissue bearing mycelium and oospores were placed on pepper fruits still attached to the plant and the whole was covered with a moist chamber for 48 hours. No infection occurred. A watch glass containing pieces of infected tissue forming oospores in water was so placed that the blossom ends of fruits hung suspended in the water for two days. Even when the fruit had been pricked with a needle, no infection resulted from this treatment.

Failure of infection by this method was possibly due to the fact that no zoosporangia had been formed and no zoospores were present to find their way into the wound. When bits of mycelium are forced through the epidermis into the tissues of the pericarp, a rapid growth of the fungus and consequent decay of the fruit ensues. Successful inoculations were made also by the latter method on the variety *C. annuum longum*.

The fungus causing the rot of pepper fruits described above is believed to be *Pythium de Baryanum* Hesse. A comparison of the pepper organism with *P. de Baryanum* as described by Atkinson (1) reveals sufficient similarity to substantiate the conclusion that they are one and the same species.

The result of the attempts to produce artificial inoculation taken together with the circumstance that natural infection has been found on fruits not more than six or eight inches away from the ground indicates that infection is accomplished naturally by zoospores splashed from the ground by rain and that they enter through wounds produced by insects. Search was made of the plants for insects which might be responsible for wounds on the fruit, but none were found.

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RUST OF ONION FOLLOWED BY A SECONDARY PARASITE

J. C. WALKER

WITH TWO FIGURES IN THE TEXT

The occurrence of an aecial stage of rust was noted on leaves and seed stems of top onion (*Allium cepa* var. *bulbellifera*) at Madison, Wisconsin, on June 17, 1920. The lesions were scattered, light yellowish in color, roughly circular to oblong in shape with distinct margins, and varying from one to twelve millimeters in diameter. See figure 1A. The surface of the diseased area was slightly raised. The aecia appeared as golden yellow, raised spots which eventually rifted the epidermis. The margins of the mature sori were incised and slightly

reflexed. The aeciospores were orange to yellowish, angular, smooth-walled, with granular contents, giving them the appearance of being verruculose; 17–22 ×19–24μ. Specimens submitted to Dr. J. C. Arthur were diagnosed as probably a case of *Puccinia asparagi*, crossing to onion, which supposition was supported by the fact that the two infected onion plots were separated by a patch of asparagus. No aecial form was noted on the latter at the time, which was to be expected from

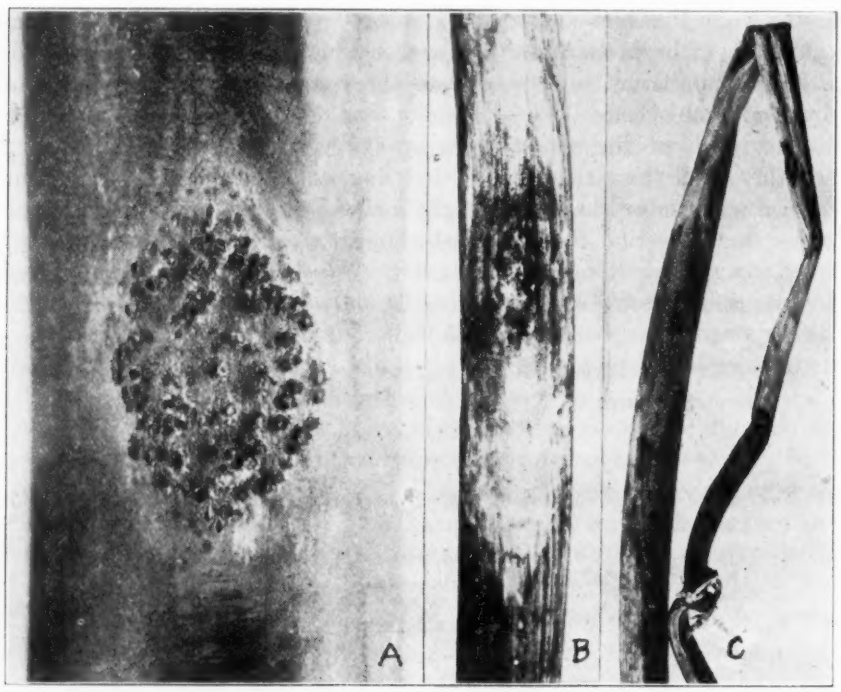

FIG. 1. *A*. Onion seed stem bearing lesion of aecia of the asparagus rust, *Puccinia asparagi* ×6. *B*. Botrytis sporophores appearing on the aecia × ¾. *C*. Onion stem finally girdled by the Botrytis × ½.

the fact that the crop had been cut closely up to that date. Late in the season, however, (September 16) many of the asparagus seed plants bore an abundance of uredinia and telia of *Puccinia asparagi*. The above description of the aecial stage on onion agrees closely with that of the asparagus rust. Sheldon (2) has successfully crossed the asparagus rust to onion, producing all three stages on the latter.

Collections of aecia on winter or top onions have been previously made by Holway (1) and by Sheldon (2) during the latter part of May

and in June. In each instance asparagus was growing nearby. The only other aecial form reported on onion and answering to this description is that of *Puccinia porri* Wint. According to Tranzschel (3), however, *P. porri* is a Hemi-puccinia; he sowed the sporidia on Allium and obtained uredospores direct.

On June 27 a re-examination of the infected onion plants showed invasion by a secondary organism by way of the rust sori. Light grey, sunken, linear lesions, irregular in outline but with definite margins, surrounded many of the rust pustules. Oftentimes these appeared before the sori were open and in consequence the aecia failed to mature. In every case, the remains of a rust pustule was found in the center of such lesions. See figure 2 and 1 B. The new lesions continued to enlarge quite rapidly and following a period of rainy weather sporulation characteristic of Botrytis appeared invariably on the dead tissue. Eventually, nearly every rust lesion found in the field showed evidence of invasion by Botrytis, and no infection by this fungus other than by way of rust sori was noted. The seed stems were eventually girdled, resulting in the upper portions falling over. See figure 1C. The actual loss in this case was not serious since the top sets were nearly mature before many stems were girdled. Should such an attack occur upon seed plants of the common onion, however, considerable damage might result. A pure culture of the Botrytis form was obtained and wound inoculations into white onion bulbs were made. No signs of decay resulted, indicating the species to be different from those causing onion neck rot. Apparently it is capable of invading only as a secondary organism. Specimens and cultures have been preserved for identification and further study.

FIG. 2. *A*, *B*. Rust lesions on onion seed stems invaded by *Botrytis* sp. Note the small, light-colored, sunken spots in the rust pustules × ¾. *C*. The linear lesion developing around an immature rust pustule × ¾.

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NOTES ON THE CITRUS-ROOT NEMATODE, TYLENCHULUS SEMIPENETRANS COBB

L. P. BYARS

WITH ONE FIGURE IN THE TEXT

During the years 1914 to 1917, inclusive, the writer, while prosecuting active investigations on various nematodes attacking plants, had opportunity to make certain minor observations and studies on the nematode *Tylenchulus semipenetrans* Cobb, which parasitizes the roots of citrus species. These consisted mainly of observations on its distribution in the state of Florida, of studies of the symptoms of the disease both on the top and roots of citrus trees of various kinds, and of certain preliminary experimental work on control. The results were sufficient to indicate that the organism may prove to be a serious factor in connection with the citrus industry. It seems desirable to record the results of these incidental studies at this time, since the writer is contemplating a change of duties which will not permit of further work upon this parasite. Further experimentation appears to be important.

Thomas (5) in 1913 made the initial report of the occurrence of this nematode in California. The parasite has since been discovered on citrus from many parts of the world. Cobb (2) in 1914 found it on citrus material collected from one point in Florida and from California, Spain, Malta, Palestine, and Australia. In 1915 Trabut (6) reported the nematode on collections from Algeria, and advocated control methods involving the use of chemicals. In so far as is known by the writer, it has not been reported to occur in other countries.

DISTRIBUTION

At one time or another the writer has examined trees in all of the main citrus growing sections of Florida for nematode infection, and has found it at only three points, namely: Glen St. Mary, Gainesville, and Brooksville. The infection at Glen St. Mary was observed on 10- to 15-year-old grapefruit trees growing in a variety test on a well drained, dark, loamy soil. At Gainesville it was found to a slight extent on a few 6- to 8-year-old orange trees growing on very light sandy soil in the test grove of the Florida Agricultural Experiment Station. Severe infection was discovered on nearly all of a large variety of citrus plantings being tested at Brooksville, Florida, at the Plant Introduction Field Station of the U. S. Department of Agriculture. The trees were growing on a poorly drained loam soil—so-called hammock land—and as a rule were not in a healthy vigorous condition. Thus it seems that the nematode does not occur generally over the citrus sections of Florida, as is the case in California according to the report of Thomas (5). Its occurrence at these three points in Florida is significant in view of the fact that at each of these places there has been considerable introduction of citrus stock from other states and even from foreign countries.

In 1917 the writer was enabled, through the courtesy of members of the Office of Seed and Plant Introduction of the U. S. Department of Agriculture, to make what seems to be a significant discovery of the nematode. It was found on the roots of citrus seedlings which had been collected in Brazil by Dorsett and others (3). Some of these seedlings were tangerines, *Citrus nobilis deliciosa* (Tenore) Swingle, but the majority were navel oranges, *Citrus sinensis* (L.) Osbeck, or the species of citrus, "white selecta," from which our Washington navel oranges originated. It therefore seems quite possible that the nematode may have been originally introduced into the United States from Brazil along with early importations of citrus stock.

APPEARANCE OF AFFECTED TREES

Thomas concluded after making examinations of a large number of trees in California that, with few exceptions, a mottling of the leaves accompanied nematode infection and that the nematode very probably was an important factor in causing malnutrition, mottling, or deterioration of many citrus groves. The writer's observations differ somewhat from these in that he has been unable to find on the aerial parts of infected trees definite signs of disease which could be used as reliable criteria for the presence of the nematode on the roots. An abundance

of the parasites have been noted on both young and old trees which showed severe frenching, leaf mottling, dieback, general loss of vigor and growth, or other troubles of this nature; and likewise the organism has been found in great numbers on trees which presented none of these abnormal appearances and which were apparently vigorous and healthy. Furthermore, nematode-free trees growing in badly infected groves were discovered that exhibited to a marked degree one or more of the pathological symptoms just mentioned. These conflicting results were secured from the microscopic examination of a large number of representative trees on the Plant Introduction Field Station of the U. S. Department of Agriculture at Brooksville, Fla., and in other groves. That the organism is an active injurious parasite there can be little doubt, but the writer thinks there is still lacking observational and experimental evidence to show clearly its symptomatic effect on the aerial portion of trees.

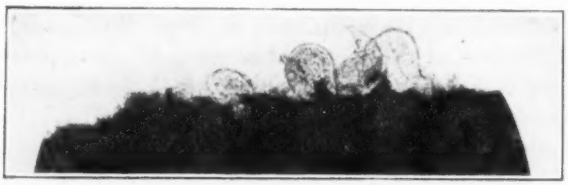

FIG. 1. PHOTOMICROGRAPH SHOWING A GROUP OF ADULT AND DEVELOPING NEMATODES ON THE FIBROUS ROOT OF A CITRUS SEEDLING IMPORTED FROM BRAZIL. × 60

On the root system of affected trees it is likewise difficult or impossible to detect sure signs of infection unless the nematodes themselves are seen. An abundance of the parasites were found on roots which appeared to be sound and normal. Moreover, few of the nematodes could be found on old dead roots. This indicates that perhaps as with other parasitic nematodes the organism leaves the host tissues as soon as the latter die. Infection can be determined with certainty only by detection of the causal nematode *Tylenchulus semipenetrans*, larval and adult stages of which may be readily seen with the aid of a hand lens adhering to the fibrous roots. In figure 1 is shown a characteristic group of adult or developing nematodes attached to the root by means of their anterior ends which are embedded in the tissues. While such adults can be seen even with the unaided eye, the writer has found that, by placing suspected roots in a Syracuse dish of water and examining with a dissecting binocular microscope a rapid and much more satisfactory examination of the roots can be made.

The citrus nematode in the adult stages is not likely to be mistaken for other nematodes. So far as the writer's observations go, no other nematode parasitizes citrus. However Neal (4), who in 1889 worked in Florida on the common root knot nematode, *Heterodera radicicola* (Greef) Müller, claimed that this organism produced galls on the roots of citrus. The writer has repeatedly tried without success to infect in greenhouse and field various species of citrus with the root-knot nematode, and has also made a large number of examinations of citrus roots growing in the field beside roots of other plants which were severely infected, without ever finding them parasitized. Bessey (1) and other investigators report similar observations.

HOT WATER TREATMENT

Favorable results secured by the writer from the use of hot water in killing the root-knot nematode when embedded in the tissues of certain tuberous crops suggested the possible application of this means to the eradication of the citrus nematode from the roots of seedlings or young trees. Consequently, on June 25, 1915, at Brooksville, Fla., the writer treated, in lots of three, the roots of badly infected young citrus trees by placing them in water at different temperatures for various lengths of time, as follows: 110° F. for 20 min.; 120° F. for 15 min.; 125° F. for 15 min.; 130° F. for 10- and 15-minute periods; 135° F. for 10 min.; 140° F. for 10 min. In addition three severely infested trees were placed in tap water at room temperature for 25 min. and used as a check. All seedlings were set out immediately after treatment in the open in isolated plots on non-infested land. A final examination of the roots of the trees was made on August 10, 1916. An abundance of the citrus nematodes was found on the check trees and on those treated at 120° F. for 15 min. but none could be detected on those treated at any of the higher temperatures. Those trees treated at 135° and 140° F. respectively were dead and the others for the most part showed ill effects from the treatments. This, however, is not surprising, as the hot water submersion was given in midsummer at a time when the trees were actively growing. In winter they could very probably withstand much higher temperatures and longer exposures without injurious effects. The results of this single limited experiment indicate that there may be practical possibilities in hot water treatment for the elimination of this pest from nursery stock.

BUREAU OF PLANT INDUSTRY
U. S. DEPARTMENT OF AGRICULTURE
WASHINGTON, D. C.

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THE TRANSFERENCE OF POTATO LATE BLIGHT BY INSECTS

W. J. MORSE

Records of undisputed transference of the spores of parasitic fungi by insects are by no means lacking in phytopathological literature. As a rule, however, these represent cases where the spores are more or less resistant to drying. The conidia of the potato late blight fungus, *Phytophthora infestans* de Bary, being thin-walled and very delicate, hardly belong to this class. Undoubtedly most pathologists with considerable field experience with late blight would agree that under favorable weather conditions it is probable that certain insects play an important role in transferring the disease between adjoining plants and in distributing the conidia from one portion of an affected plant to another. The writer, at least, has always thought it improbable that late blight is carried any distance in this way from field to field. The following observations suggest that under especially favorable conditions such may be the case.

A small greenhouse is maintained in connection with our plant disease work. During a considerable portion of the year it is used largely for growing potatoes for experimental purposes. No greenhouse studies have ever been made on late blight there and no potatoes affected with late blight rot have ever been planted in this house. With one exception the plants have always been free from this disease.

Early in September, 1915, the house was practically filled with rapidly growing potato plants. One of these, located near an open win-

dow on the south side, developed a severe case of late blight, and the disease spread rapidly through the house. It was noted that the flea beetle, *Epitrix cucumeris* Harris, and the pink and green potato aphid, *Macrosiphum solanifolii* Ashmead, were by no means uncommon on all of the plants.

Weather conditions had been very favorable during the previous eight weeks for the development of blight in the fields. There were 13 rainy days each in July and August, or 26 out of 62. During this period the total rainfall at Orono was 11.34 inches. The only growing potatoes within half a mile or more was a small plot of several varieties located about 150 yards northeast of the greenhouse. An examination showed that these plants were in the last stages of destruction by late blight, but the causal fungus was still fruiting abundantly on those leaflets which were partially alive.

The greenhouse had no open ventilators or windows facing toward this plot of blighting potatoes, moreover taller buildings, lying between, still farther prevented the possibility of the spores of the blight fungus being carried by wind from the field to the greenhouse. Flea beetles were present in great numbers on the potato plants in the field. The aphids were less plentiful, but were numerous. These facts suggested that the blighting field mentioned was the source of the infection in the greenhouse and that the insects in question acted as carrying agents.

Conditions prevented making an actual demonstration that the disease could be communicated from diseased to healthy plants by transferring the insects from one to the other, for no plants were available which had no possibility of not being already infected. An attempt was made to determine if the conidia of the fungus could be demonstrated by microscopic examination as adhering to the bodies of the insects. A large number of both species were examined in this way, both from the greenhouse and from the field. No spores were found on any of the insects that came from the greenhouse and none on the plant lice from the field, but an occasional one could be found adhering to the bodies of the flea beetles. Twenty or more flea beetles, collected from plants in the field, were placed in each of a number of small vials. The vials were partly filled with distilled water and the mixture of insects and water vigorously shaken. Then the liquid was, either with or without the insects, transferred to a small centrifuge tube and centrifuged for several minutes. An examination of the sediment at the bottom of the tube in a number of, but by no means all, cases, showed one or two conidia of the late blight fungus.

While circumstantial evidence strongly indicated that either the flea beetles or the potato aphids were the responsible agents in carrying the disease from the field to the greenhouse, positive proof of this was not obtained. No positive evidence was obtained that the spores of the fungus adhered to the bodies of the aphids. On the other hand it is apparent that the flea beetle may act as a carrier, but this is by no means the universal rule where they are taken from plants on which an abundance of the conidia of the late blight fungus are being produced.

MAINE AGRICULTURAL EXPERIMENT STATION

LIGHTNING INJURY TO POTATO AND CABBAGE[1]

C. R. ORTON

WITH ONE FIGURE IN THE TEXT

POTATO

The writer had an opportunity at Girard, Pa., in 1919, to observe carefully a field of Petoskey potatoes which had been struck by lightning. The injured patch was first observed by Mr. Mohring, the owner, on August 22, after a severe electrical storm two days previously, but it was not until August 29 that the following observations were made:

The area which involved about 125 plants was nearly circular in outline, being about thirty feet in its greatest diameter. When observed by me, all the plants in the center of the area were killed—those nearer the margin were nearly dead and the injury became less as the plants were further removed from the center. The stems of the more severely affected plants were collapsed from near the ground upwards for several inches and often nearly to the tip. At this time the cortex was whitened where it had not already become overgrown with saprophytic fungi. The pith at and just above the ground was hollow and white. The greener portions of the stems showed a flattened or compressed condition with the pith browned and the xylem region sometimes brown. The pith from the ground to the attachment with seed piece was not hollowed but was usually browned to within a short distance from the old seed piece. The pith in some of the lateral branches was also browned for a short distance from the nodes.

[1] Contribution from the Department of Botany, The Pennsylvania State College, No. 28.

The severest injury appeared two or three inches above the ground where the stalk was entirely dead and sometimes, though not always, split. Occasionally a stalk in an affected hill seemed to escape the injury. It was noticeable that when such a condition was found the largest stalk was the one which always showed the most pronounced injury.

It should be noted that these observations were made nine days after the injury took place and the symptoms exhibited were undoubtedly in the later stages.

CABBAGE

No reference has been found to lightning injury on cabbage, although Professor L. R. Jones reports in Phytopathology **7**: 140–141, 1917, a case of lightning injury to kale occurring in Michigan.

On July 29, 1920, while passing through Erie, Pa., I visited the office of S. S. Lehman, County Agent, and was shown some cabbage plants which had been brought to him the day before from near Edinboro, Pa. The grower reported that a circular spot had appeared suddenly in the field and seemed to be spreading. An examination of the plants immediately suggested to me the probability of lightning injury and arrangements were made to visit the field. This was done on August 1.

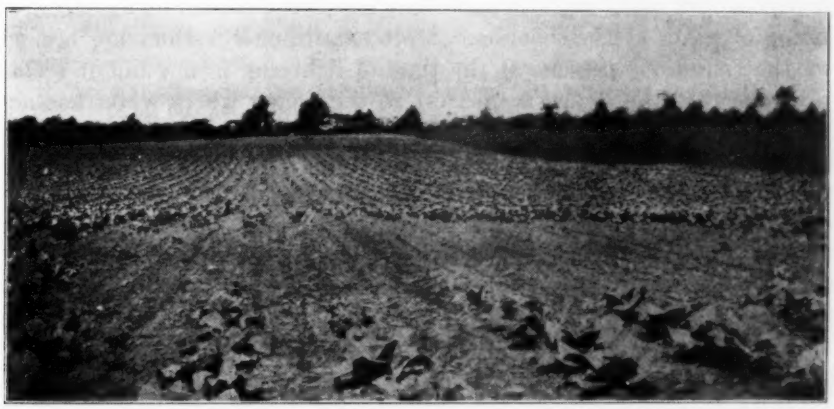

FIG. 1. CABBAGE FIELD SHOWING AREA KILLED BY LIGHTNING
Photographed by courtesy of S. S. Lehman

The cabbage field consisted of about fifteen acres in which the stand was very uniform except in two or three low spots where the plants had been drowned by the frequent rains.

The spot in question was upon the higher, better drained portion of the field, was almost circular in outline and about thirty-five feet

across, involving eleven rows. At this time all the plants in the center of the area were dead and shriveled up. The plants near the margin showed progressive stages of the injury and a rather careful examination was made. See figure 1.

The most noticeable injury occurred about two to three inches above the ground where the stems were shriveled on the more seveerly injured plants. On the plants showing lesser injury, dark purplish spots were produced upon the stem involving those portions just above and about the lower leaf bases. In some cases these purplish spots had faded and become light brown. In almost every case the mid-veins of the lower six or eight leaves showed marked injury on both sides, taking the form of collapsed, shriveled tissue.

No evidence was seen of any vascular discoloration. Aside from the noticeable injury upon the stems and mid-veins the most characteristic symptom appeared to be the destruction of the pith from a point just below the origin of the "head-leaves" nearly or quite to the ground. There was little discoloration of the region around the pith but there seems no doubt about the injury bringing about this very pronounced effect. The delicate structure of the pith possibly accounts for its striking destruction.

It seems probable that the very characteristic necrotic areas involving the cortex about the leaf-bases may have been caused by an accumulation of water at these points. More careful observations are needed on the conditions present at the time of lightning injury but it seems probable that the injury is greatest at the points where water has accumulated. This would account for the severe injury on the mid-veins and stems just above the ground—and would also perhaps account for the frequent absence of injury to the portions below ground. More observations are needed on the effect upon the pith of plants injured by lightning, but in the several cases observed by me upon potatoes and the one case upon cabbage it was most pronounced and may prove to be a help in diagnosis.

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PHYTOPATHOLOGICAL NOTES

The mucilage of mistletoe berries as an adhesive.—It is not generally known that a very effective adhesive may be made from the mucilaginous or viscid endocarp of the berries of Phoradendron, Viscum, and related genera. A thin paste may be prepared by beating up the berries in water. The residue from the crushed fruit may then be strained off and the solution allowed to evaporate to the desired consistency. A low alcohol, phenol or some other preservative that will not cause coagulation, may be added. This preparation will keep indefinitely, and has been used by the writer in herbarium work, and in many cases was found to be more effective than the ordinary library paste of the trade. It is possible that the preparation may be found of use in microscope and microtome technique. An adhesive may also be extracted from the berries of Razoumofskya species.

Many species of Phoradendron in the south and southwest produce berries in large quantities. In some sections Razoumofskya species are sufficiently abundant to admit of the collecting of berries in considerable quantities.

The adhesive nature of the mucilaginous content of these berries, especially in the case of the Phoradendrons, is probably of considerable commercial importance, and is worthy of further investigation.—JAMES R. WEIR.

Polyporus dryadeus (Pers.) Fr. on conifers in the Northwest.—*Polyporus dryadeus*, the cause of a well-known heart rot in various dicotyledonous trees, especially oaks and poplars, occurs rather frequently on conifers in the Northwest. The fungus delignifies the heartwood of the base and roots of living trees and may be the cause of windfall. The fungus most frequently occurs on *Tsuga heterophylla* and *T. mertensiana*, but it has also been collected on *Abies grandis* and *Picea engelmanni*. Collections have been made on conifers from Washington, Oregon, Idaho and Montana. The fungus is also found on various species of oaks and maples in the West. This note verifies the report made by W. H. Long (Jour. Agric. Research **1**: 247, 1913) on specimens of *Polyporus dryadeus* collected by C. J. Humphrey on *Tsuga heterophylla* in Washington.—JAMES R. WEIR.

Septoria Negundinis Ellis & Ev. in Zion National Park.—On July 22 of the present year the writer visited Zion National Park, Washington County, Utah. Upon arriving at the Wiley camp, one is impressed with the immense box-elder trees which form a sort of a lane in front of the park. At the time of the visit, however, apparently every loaf of every tree was affected by what subsequently was determined as *Septoria negundinis* Ellis & Ev. The trouble was locally attributed to the effect of a late frost which had been unusually destructive. Some of the spores of the Septoria are more than three-celled.—A. O. GARRETT.

The Imperial (British) Bureau of Mycology (to which a reference was made in Phytopathology, 9, p. 265, June, 1919) has now been established at Kew, where it will work in close coöperation with the Director and Staff of the Royal Botanic Gardens. Dr. E. J. Butler, until recently Joint Director of the Agricultural Research Institute, Pusa, and Imperial Mycologist (India), has been appointed Director, and has commenced work at the headquarters of the new bureau, No. 17, Kew Green, Kew, Surrey, England. It is intended to set up a central agency for the accumulation and dissemination of information on all matters concerning the diseases of plants caused by fungi, and for the identification of specimens, on behalf of workers in the overseas parts of the British Empire. There are over fifty appointments of professional mycologists or plant pathologists now sanctioned in the overseas Dominions, Colonies and Protectorates, and in many cases the work is carried on under conditions of considerable isolation. It will be the aim of the Bureau of Mycology to serve as a link between these workers and those in other parts of the world, and to become a central clearing house for the good of all. Specimens received for identification will be dealt with in part by the Bureau (where culture work on pathogenic fungi will be carried on) and in part by distribution to specialists in particular groups. Information will be circulated, if funds permit, by means of an abstracting journal on the lines of the "Review of Applied Entomology," and also by direct correspondence. Separates of original papers will be lent to isolated workers, and the Bureau is issuing an appeal to be supplied with two copies of separates from all who can spare them, to provide for this part of its activities. The need for some central organization on these lines has long been felt by workers in many British Colonies, and the Bureau, which is supported entirely by contributions from the Colonies, has been established to meet a definite demand.—E. J. BUTLER.

The Phytopathological Society of France.—Believing that the Plant Pathologists of America may be interested in a few facts regarding the

Phytopathological Society of France, I am offering the following brief statement based upon information given to me by M. Etienne Foëx upon the occasion of his visit here this past summer. The Société de Pathologie Végétale de France was founded in May, 1914. The first president was Professor Mangin; the first secretary general was M. Hariot; the second secretary general was M. Arnaud. The president for 1921 is Professor Viala; the secretary general for the same year is Etienne Foëx. The association issues a publication monthly in four fascicles under the title, Bulletin de la Société de Pathologie Végétale France. The society meets once a month at 63 rue Buffon, Paris Ve. The Association includes both plant pathologists and entomologists, and papers on both plant pathology and entomology appear in the Journal. The price of this Journal including membership in the Association is 12 francs. Non-members pay 14 francs per year. Anyone interested in the society or the Journal should address inquiries to M. Foëx at the Station de Pathologie Végétale, 11 bis rue d' Alesia, Paris (14e).—H. H. WHETZEL.

List of literature on phytopathology.—In view of the fact that this list is to be omitted from the current numbers of Phytopathology, the mimeographed list of current botanical and pathological literature issued every two weeks from the Library of the Bureau of Plant Industry will be sent to members of the Phytopathological Society requesting it. This list is a coöperative undertaking of the Library and the Office of Economic and Systematic Botany of the Bureau of Plant Industry. Miss A. C. Atwood, bibliographical assistant of the latter office, examines the periodicals and does the indexing, while the list is prepared and issued from the Library. Where there are several persons at the same experiment station or institution, we would prefer to send the list to the library or some central office to be distributed from there to individuals. This would materially reduce the amount of addressing and mailing to be done from Washington. Will all persons who wish to apply for the list, or who wish information concerning it, write to Miss Eunice R. Oberly, Librarian, Bureau of Plant Industry, Washington, D. C.—E. R. OBERLY.

Biography and portrait of Dr. F. Kolpin Ravn made available.—Arrangements have been made by the departments of plant pathology, Cornell University and the University of Wisconsin, with the Office of Cereal Investigations coöperating, to furnish practically free, biography and photograph of the late Dr. F. Kolpin Ravn, of Denmark. The biography is an English translation by Mrs. Ravn

from the original by Carl Ferdinandsen published in Danish. Requests for the biography and photograph should be made to Dr. A. G. Johnson, College of Agriculture, Madison, Wisconsin, with ten cents in stamps enclosed to cover postage and wrapper.

Personals.—Mr. E. J. Wortley has resigned his position as Director of Agriculture in Bermuda to accept a similar position in Nyasaland. Mr. Wortley has made extensive studies of leaf-roll and related diseases of the potato. He is succeeded in Bermuda by Mr. E. A. McCallan, a native Bermudian and graduate of the Ontario Agricultural College, who has been First Assistant in the Agricultural Station in Bermuda since 1913.

Mr. Paul V. Siggers, Scientific Assistant, Office of Investigations in Forest Pathology, has resigned to accept the position of Pathologist with the United Fruit Company. He will be stationed at Changuinola, Panama, mail address, Box 3, Bocas del Toro, Panama. He will investigate diseases of the cocoanut palm and cacao.

Mr. Rush P. Marshall, Pathological Inspector, Office of Investigations in Forest Pathology, has accepted a similar position with the Federal Horticultural Board. He will be engaged in the work on potato wart.

ABSTRACTS OF PAPERS PRESENTED AT THE FOURTH ANNUAL MEETING OF THE PACIFIC DIVISION, AMERICAN PHYTOPATHOLOGICAL SOCIETY, SEATTLE, JUNE 17–18, 1920.

Preliminary experiments on injury to wheat from seed treatment in Washington. GEORGE L. ZUNDEL

Samples of wheat from a large number of localities in the Pacific Northwest, especially from Washington, were treated with a standard bluestone solution (1 lb. to 5 gal. water) for ten minutes. The resulting seed injury varied from 12 to over 60 per cent. Hand-threshed wheat was injured scarcely at all. When treated with formaldehyde solution, 1–40, the injury ranged from 8 to 80 per cent. The injured seed varied with the seed lot of a given variety and with the manner in which the seed was threshed. When the high speed cylinder machines are used very dry grain sustains numerous small cracks that allow the fungicide to enter to the germ and cause injury. Wheat dipped in a lime solution for 3–5 minutes after treating with either bluestone or formaldehyde sustains little injury. In each of 5 field tests beneficial results were obtained by the use of the lime bath. The use of lime after formaldehyde gave slightly better results in counteracting injury than when it followed the bluestone treatment.

Physiological studies of the effects of formaldehyde on wheat. W. M. ATWOOD

The question of the actual entry of formaldehyde has been met and apparently answered in the affirmative by microchemical tests and by measurement of the imbibitional behavior of seeds in formaldehyde solutions. The principal studies so far have been directed towards a determination of the effects of formaldehyde of varying concentration upon the respiration of the wheat as measured by carbon dioxide output. Studies extending over two seasons seem to have established the fact that there is a marked depression of respiratory rates for wheat treated to formaldehyde at a concentration of one part to eighty parts of water. This depression in respiratory rate as compared with the check is apparent, though decreasingly so, with a lowering of the concentration just noted to 1–160, 1–240, and 1–320 the strength usually used in seed treatment. At 1–400 and 1–1000 the differences between checks and treated samples were not great enough nor sufficiently constant to indicate any marked effect on metabolism. Preliminary studies of the respiratory rates of seedlings from treated and untreated seed seem to show a marked effect of the formaldehyde in slowing down metabolism subsequent to germination. The studies will be continued the coming season.

Relation of spore load to the per cent of stinking smut (Tilletia tritici). F. D. HEALD

Artificial smutting was accomplished by adding 0.005 to 3 grams of smut to 100 grams of wheat. These amounts gave a spore load of 400 to 183,000 spores per grain. Maximum smutting was obtained with a spore load of 65,000–100,000 spores per grain. The spore load was determined by microscopic counts with a Levy cell. A spore load of 533 per grain produced 9.5 per cent of smut in Jenkin's Club, but a similar load for Marquis produced no smut. Farm infected seed planted in the spring does not smut

as much as winter wheat carrying an equivalent load. This study has made it possible to examine samples of wheat and predict with reasonable accuracy the amount of smut which might appear in the crop if no seed treatment is practiced. It has also shown that much spring wheat is treated when the spore load is so low as to give a negligible amount of smut.

Observations upon the bacterial blight of field and garden peas in Montana. HARRY MILLIKEN JENNISON

The most serious disease of seed peas grown in southwestern Montana is the bacterial blight caused by *Pseudomonas pisi* Sack. (*Ps* 211. 2322033). It is conservatively estimated that this disease was responsible for a 25 per cent reduction in the yield of the 1918 crop. This blight may be expected to be present and to cause more or less severe losses during any average summer. All parts above ground are subject to attack by the pathogene but special significance attaches to pod lesions since the seed are often contaminated in affected pods. Dissemination of the disease is thought to be due largely to contaminated seed. Under favorable weather conditions spread of the infection in the field is rapid and scarcely controllable. Differences in susceptibility to attack by the blight have been noted in about 40 varieties of garden and field peas. Alaska is one of the most susceptible.

Bacillus atrosepticus van Hall, the cause of the blackleg disease of Irish potatoes. HARRY MILLIKEN JENNISON

Frank isolated and described *Micrococcus phytophthorus* as the cause of black-leg of potatoes. More recently, at least four different "species" of *Bacillus* have been described as causing the disease in question, i. e., *Bacillus atrosepticus* by van Hall in Holland (1902) *B. phytophthorus* by Appel in Germany (1903) *B. solanisaprus* by Harrison in Canada (1906) and *B. melanogenus* by Pethybridge and Murphy in Ireland (1910).

The disease attracted the writer's special interest in 1913 when a study of it as it occurred in Montana was begun. During the next two years about forty isolations of the black-leg pathogen were made, with the idea of getting at the relationship of the parasite. Later subcultures of van Hall's, Appel's, Harrison's, and Pethybridge and Murphy's organisms together with the strains of the black-leg organism isolated in Maine were obtained. In 1915 an exhaustive study of the comparative morphology and physiology of the parasites was begun at the Missouri Botanical Garden. Twelve different strains selected from those above mentioned including those of the four authors referred, to earlier, were studied. These studies were largely completed without any knowledge of what Dr. W. J. Morse had done along similar lines and before the appearance of his paper in 1917. His studies of the "species" in question lead him to conclude that they are not specifically different. As a result of the writer's investigations it appears correct to conclude that the several strains studied are specifically identical, and because of priority van Hall's name should stand. A revised description of *Bacillus atrosepticus* van Hall yields the group number 221.1113033.

The skin spot (Oospora pustulans) of the Irish potato. F. D. HEALD

This disease recently studied in England was found in several car loads of Gold Coin potatoes shipped to the Spokane market from British Columbia. Ninety-five per cent of the tubers showed lesions. Young spots show as circular, brown spots a few mm. in diameter. With age they darken, appear slightly elevated but later slightly

depressed. Under conditions of abundant moisture the causal fungus sporulates in the old lesions. The fungus appeared identical with *Oospora pustulans* described as the cause of the skin spot in England. A quantity of tubers obtained for field test showed very poor keeping qualities. Many had the eyes killed by planting time and secondary invaders caused much deterioration. It has not yet been determined whether the disease will develop under Washington conditions.

Collar rot on apple trees in the Yakima valley. J. W. HOTSON
(Published in Phytopathology **10**: 465–486. *f. 1–15.* 1920).

Heart-rot of prune and peach in Oregon S. M. ZELLER

Many orchards of these stone fruits have been examined for heart rots. It has been found that in western Oregon a greater percentage of the existing wood decay in prune and peach trees is due to the pink-bracket fungus, *Trametes carnea* (Nees) Cooke than to all other fungi. Orchard surveys show that large pruning wounds are the most common place of infection. Two other fungi, *Lenzites saepiaria F.* and *Fomes pinicola* (Swen.) Cooke, which are also usually found on coniferous hosts very frequently cause heart-rot of peach and prune.

A spur blight of pear caused by Botrytis. S. M. ZELLER

During April and May specimens of blighted spurs of d'Anjou pear were sent in from two localities in Douglas Co., Oregon. The organism isolated from these spurs belongs to the cinerea group of Botrytis. Ten to 15 per cent of the spurs are damaged and some damage is reported on Winter Nelis and Comice pears.

Some new hosts for the Rhizoctonia disease. F. D. HEALD

This disease has been found in Klickitat County, Washington in serious form on young onions, in an affected field previously planted to potatoes. Numerous cases of the disease on strawberries have been studied from various parts of Washington.

A Cytospora canker of apple and another "die-back" fungus of interest. S. M. ZELLER

An apple canker due to a Cytospora which evidently is identical to that described by Stevens (U. of Ill. Exp. Sta. Bul. 217, 1919) occurs in various parts of Oregon. It infects living, but devitalized trees. *Cyphella marginata* McAlpine, previously reported on peach in Australia only, occurs on twigs of peach and almond in Benton and Douglas counties, Oregon.

Moldy core of the Stayman Winesap. F. D. HEALD

A study was made of the fungi found in the core cavities of Stayman Winesap apples in storage at intervals of two weeks during the winter period. Practically all apples with an open calyx canal showed a slight or a very evident growth of mold in the core. The forms isolated included two species of Penicillium, two of Alternaria, two of Cladosporium, and Hypochnus, and several species which failed to fruit in cultures. All forms failed to cause a rot in sound apples except the species of Penicillium and Alternaria. The conclusion is drawn that the presence of mold in the cores of Stayman Winesaps is no indicator of their keeping qualities, but the species that is present is the important factor.

Report on pinon blister rust in California. ELLSWORTH BETHEL and G. B. POSEY
No abstract furnished.

Two new species of Sclerotinia. B. F. DANA

The choke cherry in the Palouse country is attacked by a disease causing a leaf, twig and fruit blight. Young leaves are attacked early in the spring, and the disease often advances into the twig causing extensive blighting of the shoot. Affected leaves and shoots are covered with a gray, powdery Monilia. The fungus causing this disease is a typical Sclerotinia with the Monilia or parasitic stage on leaves and twigs, and the Sclerotinia or saprophytic stage on the fallen over-wintered fruits. The causal fungus is assigned to a new species, *Sclerotinia demissa*. Another Sclerotinia disease has been observed and studied on *Amelanchier cusickii*. This causes a leaf and fruit blight. Young or immature leaves are attacked, the margins rolling upward frequently making the leaf tubular. The Monilia stage appears on the inner protected surfaces of the rolled leaves. The diseased fruits dry up but remain hanging throughout the growing season. The fallen overwintering mummies produce large numbers of minute apothecia, 1–5 mm. in diameter. The causal fungus has been assigned to a new species, *Sclerotinia gregaria*.

A new species of Exobasidium. J. W. HOTSON.

This fungus attacks the young branches of *Vaccinium parvifolium*, apparently gaining access by some wound. The stem is slightly hypertrophied, and from this swelling spine-like processes grow out, which vary in length from 2 to 4 inches, occasionally longer. In the spring, about the middle of April in Seattle, or a little earlier in some seasons, numerous bright yellow branches which later become greenish and succulent, grow out all over the infected area. By the time they are fully developed they are pinkish at the tip. Sometimes as many as 75 to 100 of these branches appear on a single infection. As they get older they become perfectly white, due to the mycelium of the fungus growing over them. On this mycelium the basidia are formed and the large basidiospores are produced abundantly.

Pathogenicity of Rhizoctonia and the fungus as a factor in crop production. B. L. RICHARDS

Read by title.

Soil temperature as a factor in influencing pathogenicity of Rhizoctonia with special reference to the potato. B. L. RICHARDS

Read by title.

REVIEWS

An Introduction to Bacterial Diseases of Plants. By Erwin F. Smith. xxx + 688 pp., 453 illustrations. Published by W. B. Saunders Company, Philadelphia and London, 1920.

There are many unique features about this new book. It is the first treatise of its kind, a product of the author's own researches, and it fills a need that was rapidly becoming urgent.

The subject-matter is arranged in five parts: I. A Conspectus of Bacterial Diseases of Plants, dealing with host-range, susceptibility, infection factors, incubation, recovery, agents of transmission, host-reactions, and other aspects of the science. II. Methods of Research, apparatus, culture media, technic of isolation, etc. III. Synopsis of Selected Diseases, in which the type, cause, technic, and literature for fourteen bacterial diseases are given, and for each disease so treated, a laboratory outline in the form of questions and suggestions is presented. IV. Miscellaneous, notes on additional diseases, suggestions for special study, the production of tumors in the absence of parasites, speculations on the chemical and physical stimuli underlying tumor-formation, and on the production of teratosis in the absence of tumors and of parasites. V. General Observations on experimentation, interpretation, ethics of research, and similar matters.

In a word the value of this handsome volume lies in the fact that its author is an authority on the subject. It may as well be admitted that many plant pathologists do not feel qualified to carry on research in this field, and so it is gratifying that the "father of the science of bacterio-phytopathology" has put into available form the results of his many years of experience. No one will question the validity nor the cogency of the contents of this volume, and its value is enhanced by the fact that the manuscript lay in a drawer for several years after it was first written, long enough that the author had forgotten its contents, which he then revised. This procedure, as many of us already know, is distinctive of all of Dr. Smith's contributions in his special field.

The style is easy and often tremendously interesting. The book is devoid of trite expressions, laborious sentences, and various other objectionable conventionalities. On the other hand, the reader will be delighted with the conversational phrases that are well placed throughout the book. The imperative mood is very effective because the author is in position to indulge in its use, which he does with discretion.

The reviewer must disappoint those who have not seen this book by omitting the statement that "the illustrations are poor." On the contrary, they are excellent when considered from all points of view. The enlargements of bacterial colonies should prove of great service to anyone who desires to make comparative cultural studies. The casual observer may be led to inquire whether the number is not excessive, but the teacher and student alike may find in each one an impressive lesson. Figure 104 is upside down, but this error is not chargeable to the author. Figure 14 may at first appear upside down, but the plant photographed was standing on its roots and is bent downward in order to bring the diseased portion into the field.

By custom, I am constrained to refer to the fact that the pages do contain some typographical and editorial errors, but the complete list would be short. A few mistakes which the beginner should know about follow: on page 474, number 14 should

read *Bacterium* instead of *Bacillus*, number 18 *marginale* instead of *aptatum*, number 34 *tabacum* instead of *tobacum*, on page 237, figure 179A exhibits gas-formation which contradicts a statement in the text (on pages 230–231).

The book fills the need of the teacher. While it is designed primarily for laboratory use, it also contains much valuable matter for supplementary reading. The suggestions given throughout are practicable—they have been thoroughly tried—and the hints on the details of technic, such as for isolating organisms, will prove serviceable. Bacterial Diseases of Plants (its short title) is adaptable to the use of either the graduate or undergraduate student. In a few cases fuller citations to literature would facilitate the work of the teacher. The investigator will find it an indispensible manual. Problems are pointed out at every turn, and on pages 474–477 is a list of fifty-one "larger problems." Some value could have been added to the book by the inclusion of a glossary of terms, especially for new or borrowed ones such as those used in the chapter on tumor-formation.

The discussion offered in part V is of special interest. Contained therein is much that we already knew but had not hitherto expressed. Both the beginner and the mature investigator will find an inspiration in these pages.

L. R. HESLER